Amendments to the Claims:

This listing of claims will replace all prior versions, and listings, of claims in the application:

Listing of Claims:

1 (previously presented): A method for treating a subject having a lysosomal storage disease, said method comprising

administering a pharmaceutical composition to the subject wherein the composition comprises a p97 molecule covalently linked to a protein whose deficiency causes the disease and wherein the composition is delivered to a lysosome in a cell in the subject.

2 (original): The method of claim 1, wherein the subject is human.

3 (original): The method of claim 1, wherein the administering is intravenous.

4 (original): The method of claim 1, wherein the p97 molecule is human p97.

5 (original): The method of claim 1, wherein the p97 molecule is soluble p97.

6 (withdrawn): The method of claim 1, wherein the protein is α-L-iduronidase.

7 (original): The method of claim 1, wherein the p97 molecule is covalently linked to the protein by a linker from 5 to 20 atoms in length.

8 (original): The method of claim 1, wherein the linker is a polyethylene glycol.

9 (previously presented): The method of claim 1, wherein the p97 molecule covalently linked to the protein is a fusion protein of p97 and the protein.

Claim 10 (canceled).

11 (previously presented): The method of claim 1, wherein the composition comprises the p97 molecule covalently linked to the protein in a therapeutically effective amount.

12 (original): The method of claim 1, wherein the disease is selected from the group consisting of aspartylglucosaminuria, cholesterol ester storage disease, Wolman disease, cystinosis, Danon disease, Fabry disease, Farber lipogranulomatosis, Farber disease, fucosidosis, galactosialidosis types I/II, Gaucher disease types I/II/III, Gaucher disease, globoid cell leucodystrophy, Krabbe disease, glycogen storage disease II, Pompe disease, GM1-gangliosidosis types I/II/III, GM2-gangliosidosis type I, Tay Sachs disease, GM2-gangliosidosis type II, Sandhoff disease, GM2-gangliosidosis, α-mannosidosis types I/II, β-mannosidosis, metachromatic leucodystrophy, mucolipidosis type I, sialidosis types I/II mucolipidosis types II/III I-cell disease, mucolipidosis type IIIC pseudo-Hurler polydystrophy, mucopolysaccharidosis type I, mucopolysaccharidosis type II, Hunter syndrome, mucopolysaccharidosis type IIIA, Sanfilippo syndrome, mucopolysaccharidosis type IIIB, mucopolysaccharidosis type IIIC, mucopolysaccharidosis type IIID, mucopolysaccharidosis type IVA, Morquio syndrome, mucopolysaccharidosis type IVB Morquio syndrome, mucopolysaccharidosis type VI, mucopolysaccharidosis type VII, Sly syndrome, mucopolysaccharidosis type IX, multiple sulphatase deficiency, neuronal ceroid lipofuscinosis, CLN1 Batten disease, Niemann-Pick disease types A/B, Niemann-Pick disease, Niemann-Pick disease type C1, Niemann-Pick disease type C2, pycnodysostosis, Schindler disease types I/II, Schindler disease, and sialic acid storage disease.

13 (original): The method of claim 1, wherein the protein is selected from the group consisting of aspartylglucosaminidase, acid lipase, cysteine transporter, Lamp-2, α-

galactosidase A, acid ceramidase, α-L-fucosidase, β-hexosaminidase A, GM2-activator deficiency, α-D-mannosidase, β-D-mannosidase, arylsulphatase A, saposin B, neuraminidase, α-N-acetylglucosaminidase phosphotransferase, phosphotransferase γ-subunit, L-iduronidase, iduronate-2-sulphatase, heparan-N-sulphatase, α-N-acetylglucosaminidase, acetylCoA:N-acetyltransferase, N-acetylglucosamine 6-sulphatase, galactose 6-sulphatase, β-galactosidase, N-acetylgalactosamine 4-sulphatase, hyaluronoglucosaminidase, multiple sulphatases, palmitoyl protein thioesterase, tripeptidyl peptidase I, acid sphingomyelinase, cholesterol trafficking, cathepsin K, α-galactosidase B, and sialic acid transporter.

14 (original): A compound comprising a p97 molecule covalently linked to a protein whose deficiency causes a lysosomal storage disease.

15 (withdrawn): The compound of claim 14, wherein the protein is .alpha.-L-iduronidase.

16 (original): The compound of claim 14, wherein the p97 molecule is soluble p97.

17 (original): The compound of claim 14, wherein the compound is a fusion protein of the p97 molecule and the protein.

18 (original): The compound of claim 14, wherein the p97 molecule is covalently linked to the protein by a linking group which is 4-20 atoms in length.

19 (previously presented): The compound of claim 14, wherein the p97 molecule covalently linked to the protein is capable of passing through the blood-brain barrier and entering a lysosome of a cell within the central nervous system.

20 (original): The compound of claim 14, wherein the protein is selected from the group consisting of aspartylglucosaminidase, acid lipase, cysteine transporter, Lamp-2, α-

galactosidase A, acid ceramidase, α-L-fucosidase, β-hexosaminidase A, GM2-activator deficiency, α-D-mannosidase, β-D-mannosidase, arylsulphatase A, saposin B, neuraminidase, α-N-acetylglucosaminidase phosphotransferase, phosphotransferase γ-subunit, L-iduronidase, iduronate-2-sulphatase, heparan-N-sulphatase, α-N-acetylglucosaminidase, acetylCoA:N-acetyltransferase, N-acetylglucosamine 6-sulphatase, galactose 6-sulphatase, β-galactosidase, N-acetylgalactosamine 4-sulphatase, hyaluronoglucosaminidase, multiple sulphatases, palmitoyl protein thioesterase, tripeptidyl peptidase I, acid sphingomyelinase, cholesterol trafficking, cathepsin K, α-galactosidase B, and sialic acid transporter.

21-29. (cancelled)